CARDIOVASCULAR RESPONSES IN PRESSURE BREATHING

By

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Pressure breathing is an emergency measure to bring the flyer safely down to lower altitudes in the event of loss of cabin pressurisation at altitudes above 40,000 ft. At these altitudes oxygen has to be breathed at a positive pressure to prevent the effects of hypoxia. However pressure breathing besides causing physical discomfort, leads to various disturbances in the body mainly by its mechanical effects including cardiovascular changes.

The most important cardiovascular change produced by pressure breathing is the displacement of blood from the central pool to the periphery. The increased intrathoracic pressure prevents the venous return from the peripheral veins to the heart. Since the arterial inflow continues, peripheral venous distension occurs and is followed by oozing of fluids from the blood into the tissues. These changes lead to a reduction of effective blood volume. Fenn *et al.* (1947) observed a reduction in the effective blood volume by about 8-10% when breathing pressure of 30 cms. water was applied to the subject in supine position. Fenn and Chadwick (1947) recorded a venoconstriction in the fingers during pressure breathing and attributed this to a reflex initiated by the expansion of chest. Ernsting (1956) also has observed that pressure breathing causes a reflex venoconstriction in the hand veins when no counter pressure is applied. In a subsequent work Ernsting (1966) found that breathing pressure of 30 mm. and 60 mm. Hg. resulted in an immediate reduction of the effective blood volume by about 190 ml. and 300 ml. respectively, when trunk counter pressure was employed. These reductions aggravated after 5 mts. of breathing at these pressures. The reduction of effective blood volume leads to a reduced venous return, and a consequent fall of cardiac output. Fall of cardiac output with pressure breathing has been reported in dogs by William and Horwath (1959) even when counter pressure was applied with partial pressure suits. Reduction of cardiac output leads to a reflex vasoconstriction through its influence on the carotid and aortic baro receptors, with a consequent rise in mean arterial pressure. Aviado *et al.* (1951) have suggested the existence of other receptors in the low pressure region of the intra thoracic vascular bed, which also may be responsible for increase in mean arterial pressure and cardiac acceleration. We have assessed the cardiovascular responses in 400 healthy subjects by measuring the blood pressure and heart rate.

Procedure

The subjects for this study were 400 healthy young IAF Pilots who reported to the

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Institute of Aviation Medicine for medical evaluation. These pilots were medically examined and subjected to ECG and GTT before they were subjected to pressure breathing. The subjects were classified into 6 groups based on age and body weight (Table I). Each subject was given pressure breathing at 30 mm. Hg. and then at 65 mm. Hg. through a P/Q mask and a Mark 20 oxygen regulator. The durations of application of these pressures were 3 mts. and 4 mts. respectively.

TABLE I

Distribution of Subjects in the various age and weight group combinations (n = 395 IAF Pilots)

Group	Age in Years	Weight in Lbs.	Number of subjects
A_1B_1 ..	25-35	101-130	95
A_1B_2 ..	25-35	131-160	215
A_1B_3 ..	25-35	161-190	41
A_2B_1 ..	35-45	101-130	4
A_2B_2 ..	35-45	131-160	29
A_2B_3 ..	35-45	161-190	11

A recovery period of 5 mts. was allowed between the two pressures. Counter pressure to the trunk was employed through a pressure jerkin, when pressure breathing was done at 65 mm. Hg. The subjects were connected to a multichannel polygraph and were monitored for pulse, blood pressure and respiration. The pulse was recorded by the ECG electrodes through the bipolar limb leads. Blood presure was recorded by indirect method. A capacity microphone responsive to frequencies from 0.3 to 600 CPS was used to pick up arterial sounds over the brachial artery of the subject, which were recorded on one channel of the polygraph, while the cuff pressures used to occlude the blood flow in brachial artery were recorded by electrodes planted in a mercury manometer at intervals of 10 mm. Hg. each on a different channel of the polygraph. The criterion used for systolic pressure was the appearance of the first distinct wave after release of cuff pressure. The diastolic pressure was noted when the amplitude of wave showed a fall by more than 1/3 of the last one recorded. The respirations were recorded by a pneumotachograph. The pulse rate and respiration were monitored continuously throughout the time the pressure was administered but the blood pressure was recorded only in the 1st, 3rd and 4th minutes.

Results

The mean pulse rates at baseline and at different breathing pressures are given in

TABLE II

Mean Pulse Rate at Base Line and with Pressure Breathing (n = 395 IAF Pilots)

Group	Base Line	30 mm. Hg.		65 mm. Hg.		
		1 mt.	3 mts.	1 mt.	3 mts.	4 mts.
A_1B_1 ..	81.1	100.2	100.7	125.4	139.0	139.4
A_1B_2 ..	81.1	101.1	101.4	123.3	136.9	139.5
A_1B_3 ..	80.1	96.7	98.4	122.1	134.5	134.5
A_2B_1 ..	87.5	95.0	105.0	127.5	150.0	145.0
A_2B_2 ..	81.2	100.2	95.7	125.0	135.0	135.7
A_2B_3 ..	81.6	84.1	93.2	115.0	123.2	125.0
Entire Group.	81.0	100.0	99.4	123.4	136.7	138.4
	(10.4)	(18.4)	(18.4)	(23.4)	(22.9)	(23.4)

(Figures within brackets are standard deviations.)

Table II. It is seen that the pulse rate increased significantly when pressure breathing was administered; the increase was proportional to the magnitude of pressure applied. The overall increase was larger in the lower age and weight combinations, compared to the higher age and weight groups.

The mean arterial pressure at base line and at different times during pressure breathing are given in Table III. These pressures showed a significant increase above the base line in all the groups. At a breathing pressure of 30 mm. Hg., the mean arterial pressure showed an increase by approximately 70% of the extra positive pressure while at pressures of 65 mm. Hg. the pressure increased by approximately 60% only. It was also seen that the mean arterial pressure recorded in the 3rd and 4th minute were not significantly different from that at 1 minute.

Pulse pressures, although important in the study of cardiovascular responses to pressure breathing, could not be relied upon in our records since indirect B.P. recording gives higher values for systolic and lower value for diastolic due to changes in blood pressure caused by respiration while doing pressure breathing. Values of mean B.P., however, are not significantly affected as the higher value of systolic is compensated by the lower value of diastolic.

The respiratory rates showed no significant variation from their base line values in any of the groups. Since the study did not cover the respiratory flow patterns, the respiratory rates *per se* were considered to be of no significance, and were, therefore, not subjected to analysis.

A total of 5 subjects showed a tendency to pressure breathing collapse while breathing at 65 mm. Hg. pressure, with counter pressure applied to the trunk. Pressure breathing was discontinued in these subjects as soon as the end point was reached. The criteria for the end point was a sudden brady-cardia, and/or a sudden drop in arterial pressure. The pulse rates and mean arterial pressures recorded in these subjects

TABLE III

Mean Values of M.A.P. (Mean Arterial Pressure) at Base Line and with Pressure Breathing
(*n* = 395 *IAF Pilots*)

Group	Base Line	30 mm. Hg.		65 mm. Hg.		
		1 mt.	3 mts.	1 mt.	3 mts.	4 mts.
A_1B_1 ..	91.4	109.5	109.0	132.1	131.1	128.8
A_1B_2 ..	94.7	115.0	114.6	134.6	135.7	135.0
A_1B_3 ..	97.0	117.6	117.3	140.4	141.8	137.9
A_2B_1 ..	94.2	114.2	115.5	128.0	134.2	135.5
A_2B_2 ..	96.6	115.8	117.0	138.5	137.5	137.3
A_2B_3 ..	97.1	116.6	116.2	145.5	140.5	146.6
Entire Group.	94.4 (8.0)	114.0 (11.5)	113.8 (11.8)	135.2 (13.7)	135.6 (13.4)	134.4 (14.8)

(Figures within brackets are standard deviations.)

are given in Table IV. In addition to the parameters listed in this table, the subjects had profuse sweating and complained of a feeling of fainting and nausea. All these cases recovered spontaneously soon after the pressure breathing was discontinued.

TABLE IV

Pulse Rates and Mean Arterial Pressures at Base Line and with 65 mm. Hg. Pressure Breathing in 5 Abnormal Cases

Subject No.	Pulse Rate (Per mt.) 65 mm. Hg. Pressure			Mean Arterial Pressure mm. Hg. 65 mm. Hg. Pressure			
	Base Line	Reading	End point	Base Line	First Reading	End point	Time to collapse (mts.)
1 ..	84	132	60	88	116	73	2.00
2 ..	78	144	84	109	147	73	3.30
3 ..	72	132	78	90	107	83	3.00
4 ..	92	144	60	100	148	70	2.30
5 ..	84	144	96	103	158	76	3.00

Discussion

The cardiac acceleration with pressure breathing was found to be comparatively less marked in the older age and higher weight group subjects. Even though the number of subjects in this group was only 11, the deviations assume significance when the responses at two pressures and different time intervals are considered. Table II shows that the cardiac acceleration was less marked in all the recordings at both 30 mm. and 65 mm. Hg. pressures. Homeostatic mechanisms are known to deteriorate with age. It is likely that these deteriorate in obese individuals also. This aspect needs further investigation.

The mean arterial pressure showed a relatively smaller rise with breathing pressures of 65 mm. Hg. as compared to those of 30 mm. Hg. although trunk counter pressure was applied in the former. This is accounted for by the greater reduction of cardiac output due to a reduced venous return partly because of the higher breathing pressures and partly because of the absence of reflex venoconstriction in view of the counter-pressure on trunk. This counter pressure prevented greater distension of the lungs which according to Ernsting (1956) excites the reflex. The arterial pressure stabilizes after the initial fluctuations. In our series of cases stabilisation was seen to have been achieved within the 1st minute after the pressure breathing was started. This is indicated by our observation that the mean arterial pressure in the 1st minute was not very much different from that recorded in the 3rd or 4th minute.

Pressure breathing leads to collapse after sometime, depending on the breathing pressure applied and the duration of its application. The subjects reported in this paper, developed features of impending collapse between 2 and 3 minutes after pressure breathing at 65 mm. Hg. was commenced. The clinical features observed by us were typical of a vasovagal attack marked

by a sudden hypotension, bradycardia, pallor, sweating and nausea. The vasovagal attack is due to a sudden reduction of intrathoracic blood volume due to an intense arteriolar dilatation in the muscles. Sharpey Schafer *et al.* (1955) have postulated that the large pressure transients which occur in the left ventricle during systole, when the intrathoracic blood volume is reduced markedly, may stimulate receptors which are responsible for the vasovagal reflexes. The exact mechanism which leads to the sudden arteriolar dilatation in muscles is not known. Ernsting *et al.* (1960) have observed that syncope is more readily elicited when anoxia is present. This factor could be one of the contributory causes of syncope, but is not applicable in the series being reported. Severe discomfort, pain and emotional disturbances like fear or dislike for the situation are other factors which have been suggested by some workers. General constitutional disturbances like intercurrent infections and post alcoholic effects also are stated to increase the susceptibility to syncope. These factors, as precipitating causes of syncope, are not likely in our series, since the subjects refrained from drinking during the period of their evaluation at the Institute of Aviation Medicine. Further, repeated exposure to high breathing pressures on different days showed a similar response on each occasion. Sharpey Schafer *et al.* (1958) have observed that fainting may be prevented by taking a "grip of oneself". Muscular tensing and movements can keep the arterial pressure elevated by increasing the blood in the heart above a critical level. It is likely that a sudden relaxation of muscles due to whatever cause, removes the counter pressure exerted by the muscles and allows a sudden distension of blood vessels. If this is the case, contributory factors, like severe discomfort, pain and emotional disturbances could lead to a momentary distraction with the consequent general relaxation of skeletal musculature. All the subjects recovered spontaneously on cessation of pressure breathing. This observation is a natural outcome of a rapid restoration of the effective blood volume. Depressurisation is followed by a rapid fall in the central venous pressure and a corresponding increase in the pressure gradient from the peripheral veins to the right atrium followed by a rapid rise in the venous return.

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